

Muscular adaptations to resistance exercise in the elderly

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Abstract

Neuropathic, metabolic, hormonal, nutritional and immunological factors contribute to the development of sarcopenia. This loss of muscle mass associated with ageing, is a main cause of muscle weakness, but the loss of muscle strength typically exceeds that of muscle size, with a resulting decrease in force per unit of muscle cross-sectional area. Recent evidence suggests that, in addition to a reduction in neural drive and in fibre specific tension, changes in muscle architecture contribute significantly to the loss of muscle force through alterations in muscle mechanical properties. Older muscle, however, maintains a high degree of plasticity in response to increased loading since considerable hypertrophy and a reversal of the alterations in muscle architecture associated with ageing are observed with resistive training.

Keywords: Sarcopenia, Skeletal Muscle, Muscle Architecture, Ageing, Strength Training

Introduction

Sarcopenia, the loss of muscle mass associated with ageing, is an inevitable phenomenon secondary to atrophy and to motor unit loss following chronic neuropathic processes¹. From 20 to 80 years of age, about 40% of muscle mass is lost, this process becoming particularly faster after the 5th decade since 30% of muscle mass is lost between 50 and 80 years of age². In addition to the progressive denervation of motor units, mainly relevant after the 7th decade, other mechanisms that contribute to sarcopenia are metabolic, hormonal, nutritional and immunological factors¹. However, one cause of sarcopenia that is often overlooked, is the reduction of physical activity with ageing. In fact, despite the numerous studies that showed the effect of disuse on muscle atrophy³⁻⁸, the role of disuse in the loss of muscle mass associated with ageing is poorly documented. However, we have recently shown that even healthy, non-sedentary elderly individuals, aged 70+ years, are about 30% less physically active than adults in their twenties⁹. Disuse seems therefore to significantly contribute to sarcopenia, a theory also supported by

recent observations of D'Antona et al.¹⁰ who compared single fibre properties of young, active elderly and immobilised elderly subjects. These authors found that the effects of disuse on fibre force production are additive to those of ageing, indicating that in 'normal' ageing both mechanisms, i.e., ageing *per se* and disuse are operating. It is also likely that disuse is the primary cause of fibre atrophy (particularly of type II fibres) commonly found in old age since, as the present paper will show, muscle mass can be significantly improved by strength training even in old age.

Although sarcopenia is commonly regarded as a major cause of muscle weakness in old age, changes in muscle architecture associated with sarcopenia have seldom been considered as mechanisms contributing to this phenomenon. This is notwithstanding the notion that muscle architecture is a major determinant of the most fundamental mechanical properties of muscle contraction: the length-force and force-velocity relationships¹¹. Yet, we have recently shown that human muscle architecture is markedly altered in old age, since both fibre fascicle length and pennation angle of the plantar flexor muscles were found to be smaller in older compared to younger individuals¹². Also, little was known on the reversibility of these changes in response to training but some of the latest evidence obtained in our laboratory shows that elderly muscle is highly malleable in response to increased loading¹³.

Hence, the present paper will discuss the adaptations of elderly muscle to strength training with particular attention to these recent findings.

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Population	Training Duration (weeks)	Δ MVC (%)	Δ MVC /day (%)	Δ CSA (%)	Δ CSA/day (%)	Reference
Elderly (65-81 yrs)	16	19.0	0.17	7.4	0.07	26
Elderly (60-72 yrs)	12	16.7	0.20	9.3	0.11	17
Elderly (61 yrs)	10	17.0	0.25	9	0.12	19
Elderly (85-97 yrs)	12	37	0.44	10	0.11	15
Young adults	24	26.8	0.16	6.8	0.04	27
Young adults	12	15.0	0.18	5.7	0.06	28
Young adults	24	29.6	0.18	19.0	0.11	29

Table 1. Percentage gain in muscle CSA and MVC with strength training in elderly and young individuals.

Muscular adaptations to strength training in old age

Several studies have shown that the loss of muscle strength with age can be significantly reduced by strength training. In essential locomotor muscles such as the knee extensors, the increase in muscle strength, expressed as weight-lifting capacity (1 RM), ranges between 30 and 152%¹ and since it seems weakly related to training duration, it is likely to be more dependent on load intensity and initial training status of the subjects. As in young subjects, the gain in muscle strength is due both to neural as well as muscular factors¹⁴. As a matter of fact, an increase in neural drive, expressed either as activation capacity, recruitment and/or motor unit firing frequency as well as an increase in muscle cross-sectional area in response to training in old age have been found by several authors^{13,15-16}. Similar to young subjects, both type I and II fibres have been found to hypertrophy with strength training in old age¹⁷⁻²⁰ and although it has been suggested that, like in young adulthood, strength training in old age results in an increased expression of type IIa with a concomitant decrease in type IIb myosin heavy chain (MHC) isoforms²¹, other evidence indicates a decrease in the number of hybrid fibres co-expressing multiple MHCs and an increased expression of MHC-I isoform²²⁻²³.

Fibre hypertrophy following strength training in older individuals is reflected by an increase in muscle cross-sectional area (CSA) and volume and it is noteworthy that, when expressed in terms of percent increase per day of training, the

increase in muscle CSA is quite similar to that found in younger adults (Table 1). This conclusion is consistent with the observation of a similar increase in exercise-induced increase in muscle protein synthesis in frail elderly and young women²⁴.

Changes in quadriceps muscle cross-sectional area and volume, architecture and activation capacity were recently investigated in a 14-week strength training programme in a group of elderly men and women (n=9) aged 70+ years¹³. Training resulted in a 14-23% increase in weight-lifting capacity and in a 11% increase in maximum isometric fascicle force with a modest increase in activation capacity (3-6% P<0.05). Muscle anatomical CSA and volume were increased by 10% and 6%, respectively. After strength training, both vastus lateralis resting fascicle length and pennation angle were significantly increased by 8-10% (P<0.01) and 28-35% (P<0.01), respectively. From the ratio of muscle volume (VOL) to fibre fascicle length (Lf), we were able to calculate the muscle physiological cross-sectional area (PCSA=VOL/Lf). Paradoxically, since the increase in Lf was greater than that of VOL, PCSA did not actually increase after training. However, because of the significant increase in maximum isometric force, a 19% increase in force/PCSA (specific force) was found after training. This finding seems particularly important when considering the frequently reported deterioration in 'muscle quality' (force per CSA) in old age. In addition to this increase in specific force, a noteworthy finding was that optimum fascicle length increased by 11% after training, indicating a change in the length-force (L-F) relation of the muscle following these changes in muscle architecture. Most importantly, these

changes in L-F relation occurred in the presence of a remarkable increase in tendon stiffness (69%)¹³. From a functional point of view such adaptations seem very advantageous since it was estimated that the training-induced increase in fascicle length is compensated by an increase in tendon stiffness, thus enabling the muscle fibres to maintain their operational range within the optimum region of the L-F relationship²⁵.

In conclusion, elderly muscle is highly malleable to regimens of increased loading for it displays a large increase in protein synthesis leading to a significant increase in muscle mass. This training-induced hypertrophy is associated with marked changes in muscle architecture that, together with an increase in tendon stiffness, represents a very effective adaptation for counteracting the loss of muscle force associated with sarcopenia.

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